## **CLAIMS**

- 1. Use of the protein annexin A3 as a diagnostic marker for prostate cancer.
- 2. Use according to claim 1, characterized in that it is a matter of specific subtypes of prostate cancer.
- 3. Use according to claim 1 or 2 characterized in that an upregulation of annexin A3 compared with controls is investigated.
- 4. Use according to one of the preceding claims, characterized in that an upregulation of annexin A3 combined with a downregulation of annexin A1, annexin A2 and/or annexin A5 is investigated.
- 5. Use of at least one active substance which interacts with the protein annexin A3 and in particular influences, preferably inhibits the activity and/or the abundance of the protein annexin A3, for producing a medicament for the treatment of prostate cancer, preferably specific prostate cancer patient groups.
- 6. Use according to claim 5, characterized in that the active substance is an agonist, antagonist, a deficient mutant, a dominant negative mutant and/or an antisense molecule.
- 7. Use according to claim 5 or 6, characterized in that the active substance is an antibody, preferably a therapeutic antibody.
- 8. Use according to one of the claims 5 to 7, characterized in that the active substance is at least one benzodiazepine derivative, particularly BDA250 and/or BDA452.
- 9. Use according to one of the claims 5 to 8, characterized in that the activity and/or abundance of the protein annexin A3 in exosomes is influenced.
- 10. Use according to one of the claims 5 to 9, characterized in that the active substance is a small molecular compound with a molecular weight (MW) <1000 for inhibiting the ion channel activity in membranes, preferably exosomes and/or matrix vesicles.
- 11. Use of the protein mitochondrial enoyl-coenzyme A-hydratase as a diagnostic marker for cancer.
- 12. Use according to claim 11, characterized in that an upregulation of mitochondrial encyl-coenzyme A-hydratase compared with controls is

## investigated.

- 13. Use of the protein ubiquitin-isopeptidase T and/or protein-disulphide-isomerase (PDI) as a diagnostic marker for cancer.
- 14. Use according to claim 13, characterized in that a downregulation of ubiquitin-isopeptidase T and/or an upregulation of protein-disulphide-isomerase (PDI) compared with controls is investigated.
- 15. Use of the protein serum-amyloid P-component (SAP) as a diagnostic marker for cancer.
- 16. Use according to claim 15, characterized in that a downregulation of serum-amyloid P-component (SAP) compared with controls is investigated.
- 17. Use of the protein-nuclear chloride ion channel protein as a diagnostic marker for prostate cancer.
- 18. Use according to claim 17, characterized in that an upregulation of the nuclear chloride ion channel protein is investigated when compared with controls.
- 19. Use of the protein HES1 as a diagnostic marker for cancer.
- 20. Use according to claim 19, characterized in that an upregulation of HES1 compared with controls is investigated.
- 21. Use of the proteasome alpha 2-subunit as a diagnostic marker for cancer.
- 22. Use according to claim 21, characterized in that an upregulation of the proteasome alpha 2-subunit compared with controls is investigated.
- 23. Use of the protein adenine-phosphoribosyl-transferase as a diagnostic marker for prostate cancer.
- 24. Use according to claim 23, characterized in that an upregulation of the adenine-phosphoribosyl-transferase compared with controls is investigated.
- 25. Use of the protein inorganic pyrophosphatase as a diagnostic marker for prostate cancer.
- 26. Use according to claim 25, characterized in that an upregulation of inorganic pyrophosphatase compared with controls is investigated.
- 27. Use of the proteins ubiquitin-isopeptidase T and serum-amyloid P-

component (SAP) as diagnostic markers for cancer, in which preferably a downregulation of the proteins compared with controls is investigated.

- 28. Use of at least two proteins selected from the group consisting of ubiquitin-isopeptidase T, heat shock protein 27 (HSP27), heat shock protein 90 (HSP90), protein-disulphide-isomerase (PDI), mitochondrial encyl-coenzyme A-hydratase and nucleophosmine as diagnostic markers for cancer, in which there is an investigation of a downregulation of ubiquitin-isopeptidase T and/or heat shock protein 27 (HSP27) and/or an upregulation of heat shock protein 90 (HSP90), protein-disulphide-isomerase (PDI), mitochondrial encyl-coenzyme A-hydratase and/or nucleophosmine compared with controls.
- 29. Use according to one of the preceding claims, characterized in that the cancer is prostate cancer.
- 30. Use according to one of the preceding claims, characterized in that through the investigation of one or more proteins subtypes of cancer, particularly prostate cancer are diagnosed.
- 31. Use according to claim 30, characterized in that at least one protein according to claim 28 in combination with at least one protein selected from the group consisting of serum-amyloid P component (SAP), fatty acid-binding protein 3 (FABP-3), galectin, microseminoprotein beta, 14-3-3 protein beta, 14-3-3 protein zeta, nuclear chloride ion channel protein, 14-3-3 protein tau, epidermal fatty acid-binding protein (E-FABP), annexin A3, transgelin, triosephosphate isomerase and aldolase A are investigated, an investigation taking place of zero or minor modifications of SAP, a downregulation of FABP-3, a strong downregulation of galectin, a strong downregulation of microseminoprotein beta, zero or minor changes of 14-3-3 protein beta, zero or minor changes of 14-3-3 protein tau, zero or minor changes of 14-3-3 protein tau, zero or minor changes of transgelin, zero or minor changes of annexin A3, an upregulation of transgelin, zero or minor changes of triosephosphate isomerase and/or zero or minor changes of aldolase A compared with controls.
- 32. Use according to claim 30, characterized in that at least one protein according to claim 28 in combination with at least one protein selected from the group consisting of serum-amyloid P component (SAP), fatty acid-binding protein 3 (FABP-3), galectin, microseminoprotein beta, 14-3-3 protein beta, 14-3-3 protein zeta, nuclear chloride ion channel protein, 14-3-3 protein tau, annexin A3, transgelin, triosephosphate-isomerase and aldolase A are investigated, investigation taking place of a strong upregulation of PDI, a strong upregulation of HSP90, a strong downregulation of ubiquitin-isopeptidase T, a downregulation of SAP, zero or minor changes of FABP-3, a downregulation of galectin, a downregulation of microseminoprotein beta, an

upregulation of 14-3-3 protein beta, an upregulation of 14-3-3 protein zeta, an upregulation of 14-3-3 protein tau, zero or minor changes of nuclear chloride ion channel protein, an upregulation of annexin A3, a downregulation of transgelin, an upregulation of triosephosphate isomerase and/or an upregulation of aldolase A compared with controls.

- 33. Use according to claim 30, characterized in that at least one protein according to claim 28 in combination with at least one protein selected from the group consisting of serum-amyloid P component (SAP), fatty acid-binding protein 3 (FABP-3), galectin, microseminoprotein beta, 14-3-3 protein beta, 14-3-3 protein zeta, nuclear chloride ion channel protein, 14-3-3 protein tau, epidermal fatty acid-binding protein (E-FABP), annexin A3, transgelin, triosephosphate-isomerase and aldolase A are investigated, an investigation taking place of a downregulation of SAP, zero or minor changes of FABP-3, zero or minor changes of galectin, zero or minor changes of microseminoprotein beta, zero or minor changes of 14-3-3 protein beta, zero or minor changes of 14-3-3 protein zeta, a strong upregulation of nuclear chloride ion channel protein, zero or minor changes of 14-3-3 protein tau, zero or minor changes of transgelin, zero or minor changes to annexin A3, zero or minor changes of transgelin, zero or minor changes of triosephosphate-isomerase and/or zero or minor changes of aldolase A compared with controls.
- 34. Use according to one of the preceding claims, characterized in that at least one protein is detected with the aid o polyacrylamide gel electrophoresis, particularly two-dimensional gel electrophoresis, mass spectrometry, positron-radiation tomography (PRT), antibodies, ELISA, immunohistochemistry, protein chips and/or oligonucleotides, particularly the polymerase chain reaction (PCR).
- 35. Use according to one of the preceding claims, characterized in that exosomes are isolated and/or analyzed for investigating the at least one protein.
- 36. Diagnostic kit, comprising at least one substance for detecting the activity and/or abundance of at least one protein selected from the group consisting of ubiquitin-isopeptidase T, serum-amyloid P component (SAP), nuclear chloride ion channel protein, mitochondrial encyl-coenzyme A-hydratase and annexin A3 for the identification of cancerous diseases, particularly prostate cancer.
- 37. Use of at least one active substance influencing the activity and/or abundance of the proteins ubiquitin-isopeptidase T and protein-disulphide-isomerase (PDI), for producing a medicament for the treatment of cancer, in which preferably the active substance increases the activity and/or abundance of ubiquitin-isopeptidase T and/or the active substance inhibits the activity

and/or abundance of the protein-disulphide-isomerase (PDI).

- 38. Use of at least one active substance influencing the activity and/or abundance of the protein mitochondrial enoyl-coenzyme A-hydratase for producing a medicament for the treatment of cancer.
- 39. Use according to claim 38, characterized in that the active substance inhibits the activity and/or abundance of the mitochondrial enoyl-coenzyme hydratase.
- 40. Use of at least one active substance influencing and in particular increasing the activity, abundance and/or localization of the protein serum-amyloid P-component (SAP) for producing a medicament for the treatment of cancer.
- 41. Use of at least one active substance influencing, particularly inhibiting, the activity and/or abundance of the protein nuclear chloride ion channel protein for producing a medicament for the treatment of prostate cancer.
- 42. Use of at least one active substance influencing, particularly inhibiting, the activity and/or abundance of protein HES1 for producing a medicament for the treatment of cancer.
- 43. Use of at least one active substance influencing, particularly inhibiting, the activity and/or abundance of the proteasome alpha 2-subunit for producing a medicament for the treatment of cancer.
- 44. Use of at least one active substance influencing, particularly inhibiting, the activity and/or abundance of the protein adenine-phosphoribosyl transferase for producing a medicament for the treatment of prostate cancer.
- 45. Use of at least one active substance influencing, particularly inhibiting, the activity and/or abundance of the protein inorganic pyrophosphatase for producing a medicament for the treatment of prostate cancer.
- 46. Use according to one of the claims 37 to 45, characterized in that the cancer is prostate cancer, preferably specific prostate cancer subtypes.
- 47. Use according to one of the claims 37 to 46, characterized in that the active substance is an agonist, antagonist, a deficient mutant, a dominant-negative mutant and/or an antisense molecule.

- 48. Use according to one of the claims 37 to 47, characterized in that the active substance is an antibody, preferably a therapeutic antibody.
- 49. Use according to one of the claims 37 to 48, characterized in that the active substance is a small molecular compound with a molecular weight (MW) <1000 for inhibiting ion channel activity in membranes, preferably exosomes and/or matrix vesicles.
- 50. Use according to one of the preceding claims, characterized in that the active substance is at least one protein selected from the group of ubiquitin-isopeptidase T, serum-amyloid P-component (SAP), fatty acid-binding protein 3 (FABP-3), annexin A3, galectin, microseminoprotein beta, heat shock protein 27 (HSP27) and transgelin.
- 51. Use according to one of the preceding claims, characterized in that the active substance is provided in the form of exosomes.
- 52. Pharmaceutical composition comprising at least one active substance according to one of the preceding claims and at least one pharmaceutically acceptable carrier.
- 53. Method for seeking active substances for the treatment of cancer, characterized in that at least one protein selected from the group consisting of ubiquitin-isopeptidase T, serum-amyloid P-component (SAP), nuclear chloride ion channel protein, 14-3-3 protein tau, mitochondrial enoyl-coenzyme A-hydratase, annexin A3, HES1, proteasome alpha 2-subunit, adenine-phosphoribosyl transferase and inorganic pyrophosphatase and/or at least one derivative thereof is used.